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An investigation into response inhibition in distinct clinical groups within obsessive compulsive disorder

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Abstract

Response inhibition has been frequently studied in OCD with mixed results. The inconsistent findings may stem, in part, from a failure to consider the heterogeneity of disorder. This study examines behavioural and ERP components (N2 and P3) during a simple response inhibition Go/NoGo task in a sample of 48 OCD and 53 control participants. Comparisons in behavioural and electrophysiological measures were made between groups (OCD vs control), and within the OCD group itself in terms of symptoms clusters (symmetry, forbidden thoughts and cleaning) and co-morbidity status (OCD only, OCD with depression). In the OCD group the N2 component appeared more frontally localised compared to the control group. Participants with OCD demonstrated longer N2 latency and a larger difference in N2 between the NoGo and Go conditions, suggesting slower but more greater conflict monitoring. P3 had a larger amplitude in the OCD group compared to controls, indicative of greater response inhibition but it was also was reduced in the NoGo compared to the Go condition, suggesting suppressed response inhibition. No significant differences were found between symptom clusters, but those with OCD made more omission errors than those with OCD and comorbid depression. The latter also had faster P3 latencies, which combined with the behavioural data indicates slightly improved response inhibition when comorbid depression is found. Based on the results presented, it would seem unlikely that symptom clusters has contributed to previous inconsistencies, but comorbid depression could have impacted and should be considered in future research.

Background

Obsessive Compulsive Disorder (OCD), characterised by recurrent obsessions and compulsions, affects 1-3% of the general population (1-3). Although classified as a single condition, individuals with OCD have varied experiences (4). One way to characterise OCD in an individual is to use the Yale Brown Obsessive Compulsive Scale (Y-BOCS), which is considered the gold standard tool for assessing severity and symptom diversity in OCD (5, 6). Several studies have used the Y-BOCS to identify symptom clusters, including a large meta-analyses examining factor analytic studies which found that four factors explain 79% of the variance in OCD symptoms: i) Symmetry ii) Forbidden thoughts iii) Cleaning iv) Hoarding (7-9). These four clusters appear stable across the lifespan and robust to differences in factor analytic techniques of individual meta-analyses. The same four clusters have been confirmed in other studies (10, 11), although under DSM-5, hoarding became a separate classification, leaving three symptom clusters within OCD (12). As well as symptom heterogeneity, which may be identified with the Y-BOCS, individuals with OCD often have psychiatric comorbidities, most notably depression (13). Critically, symptom cluster and comorbidity are known to impact on treatment responsiveness and therefore it is beneficial to better understand this heterogeneity (8, 14-16). Despite the importance of these factors, to date, research has typically not accounted for the heterogeneity of OCD symptoms and comorbidities within study designs which may have contributed to mixed findings in some areas (17). One such area of research is response inhibition.

Several studies have identified differences in performance on response inhibition tasks in OCD, when compared with healthy control participants, using Go/NoGo tasks (18, 19), but findings are inconsistent. Behavioural measures show both faster reaction times (20) and slower reaction times in OCD (21). There are also reports of altered error rates (22), whilst

others find no differences in any measure (23-29) in OCD. Of these studies, only one (26) noted the symptom cluster and did not consider this as a variable in the analysis. In the majority of the studies, those with comorbidities including depression were excluded (20-24, 28, 29), and where comorbid depression was recorded (25-27), it was not differentiated in the analysis. The failure of these studies to take into account two important sources of heterogeneity in OCD may have in part contributed to the inconsistent findings.

Neurophysiological data from Go/No-Go tasks focuses on the Event Related Potentials (ERPs) N200 (negative deflection at frontal and central sites at 200 – 300 ms) and P300 (positive deflection at frontal and central sites at 300 – 600 ms, (30)), often considered collectively as the N2/P3-complex (31). These N2 and P3 components are associated with the early and late phases of response inhibition, respectively, and would normally be increased in inhibition conditions (30). Analysis of N2/P3 complex in OCD show inconsistent results; N2 has been reported to be both increased (22, 32, 33) and decreased (26, 27, 34) in OCD and P3 has also been reported as both increasing and decreasing in different studies of OCD (32, 33, 35, 36) or not changing at all. As with the behavioural data, only one study noted symptom cluster but did not analyse according to it (26). The majority also excluded those with comorbid depression (22, 32, 33, 35, 36) with the remaining three studies including those with depression but not accounting for this in their analysis ((26, 27, 34). This again demonstrates that key sources of heterogeneity have been neglected in previous studies.

Whilst inconsistent results may be attributed to differences in the exact task and medication status, for example, we propose that failure to consider the heterogeneity of the disorder, specifically in terms of symptom clusters and comorbid depression, may also have contributed to the mixed findings. The aim of this study was, therefore, to assess response inhibition using

the Go/No-Go task, which has previously found inconsistent results, in a manner that allows consideration of symptom cluster and comorbid depression, in addition to considering the disorder as a whole to allow comparison with previous studies. Because of the inconsistencies reported in previous work and the fact that no studies to date have been conducted using the symptom clusters as defined by Bloch et al. (9) and DSM-5 (12), we have employed two-tailed hypotheses. Specifically, we hypothesised that there would be significant differences in response inhibition between i) control and OCD participants and ii) OCD participants with different symptom clusters and comorbidity status.

Methods

Participants

Ethical approval was obtained from the institutional ethics committee (Ref HREC/2012/#1191/1) and the work was conducted in accordance the Declaration of Helsinki. All participants provided written consent to participate.

Participants (OCD = 48; Control = 53) aged 18-60 years were recruited through local advertising. All participants had completed secondary education and reported no history of brain injury or neurological disorder. Control participants were included only if they could confirm no current or previous psychiatric disorders. Additionally, ten randomly selected control participants completed the Y-BOCS to confirm subclinical scores on the YBOCS as indicated in the standard YBOCS illness classification (i.e. less than 7 out of 40) (37), as expected for a healthy population. OCD participants were included only if they had an existing diagnosis of OCD which was validated using the Y-BOCS during participant screening, resulting in a score within the clinical range i.e. over 7 out of a possible 40. The average total YBOCS score, a marker of illness severity, for all OCD participants was 20.88 ± 1.78, which

is classed as moderate OCD. In all cases YBOCS assessment was carried out by a researcher qualified and deemed competent in administering psychological assessment. A current diagnosis of comorbid major depressive disorder was reported in 58% of those with OCD participating in the study. Participants experiencing other comorbid conditions were excluded from the study. Control and OCD groups were matched for gender, age, years in education and handedness (Table 1).

Characterisations of OCD groups

As indicated above, all OCD participants completed the Y-BOCS to confirm diagnosis at the time of testing. The total Y-BOCS score also provided a measure of illness severity. Each OCD participant was allocated to one of the three clusters based on their responses to the Y-BOCS Symptom Checklist, which contains over 50 commonly reported obsessions and compulsions across several categories, allowing participants to self-identify their most prominent symptoms. The clusters were matched for gender, age, years in education, handedness, age of onset, illness duration, illness severity, medication and use of cognitive behavioural therapy (CBT) (Table 2). There were no differences between the OCD only and OCD with comorbid depression group in terms of gender, age, years in education, handedness, age of onset, illness duration, illness severity, medication and use of CBT (Table 3).

Procedure – Behavioural Paradigm

Participants completed the commonly used Visual Continuous Performance Task (VCPT; PsyTask Software for the Mitsar System (38, 39)) with Go and NoGo conditions. We opted to use a Go/No-Go measure of response inhibition because this is an area where inconsistencies have previously been found as discussed above and the task is also very simple for participants. During each trial two stimuli were presented in stimulus pairs, as is standard practice for this task. Using this paired approach allows some separation of two operations in time: preparation to receive a stimulus and preparation to make a movement (40). It is hypothesized that the ERP

in response to the first stimulus in a pair represents sensory disengagement while the ERP in response to the second stimulus, which determines whether the trial is a Go or No-Go trial, represents motor suppression i.e. a component of response inhibition. At the start of each trial a black fixation cross appeared at the centre of the screen. After 300ms, the first stimulus was presented for a period of 100 ms. This stimulus then disappeared and was replaced by the fixation cross for a further 1000 ms, before the second stimulus appeared for a period of 100 ms. There was then a 1500 ms response period. Stimulus timing and images for the two conditions are shown in Figure 1. The different stimuli were matched for size, luminance and colour. For the Go condition participants instructed to press the left mouse button as fast as possible when the second stimulus is an animal, and for the NoGo condition, participants instructed to suppress their response and not click the mouse button when the second stimulus is a plant. Trials were presented in a pseudorandomised manner to ensure 100 trials of each condition were presented. Three behavioural measurements were made: reaction time for correct responses; the number of omission errors (not pressing in a Go trial) and iii) commission errors (pressing in NoGo trial). Before the experiment participants had the opportunity to practice the task.

EEG Recording - Event Related Potential

EEG was continuously recorded using the WinEEG (Version 2.93.59) Mitsar 21 channel EEG system (38) with nineteen pure tin scalp electrodes on a preformed electrode cap (41) positioned according to the international 10-20 system (42). The reference electrodes (A1, A2) were positioned on each earlobe. A ground electrode was placed on the midline three centimetres anterior to Fz (frontal midline electrode). Impedance levels were kept under 5 kΩ. EEG was digitally recorded on a common average montage (43). Band pass Butterworth filters were set at low pass filter 0.53 Hz, high pass filter 50 Hz, and a notch filter 45-55 Hz. The data input signals were digitised at a sampling rate of 250 Hz. Independent Component Analysis

was used to identify and remove components representing horizontal and vertical eye movements. Periods involving pulse-like voltages exceeding 75μV or slow frequencies between 0 and 1 Hz exceeding 75μV were discarded by automated rejection. Electromyography (EMG) was manually removed. Raw EEG data was baseline corrected using a pre-stimulus period of 300 ms and quantified by peak amplitude and peak latency of the N2 and P3.

Data Analysis

All data analysis was conducted in SPSS and used parametric testing after first confirming the data was suitable using the Kolmogorov-Smirnov Test and measures of skewness and kurtosis. For ANOVAs where the sphericity assumption was violated data is reported for the Greenhouse-Geiser correction (44). Hypothesis 1 (Control vs. OCD): For the behavioural data, reaction time, commission and omission errors were compared using an independent sample t-test. For the ERP data, N2 and P3 were maximal at frontal central electrodes, in line with previous studies (30, 31) and, therefore, data from Fz, Cz and Pz only were selected for analysis. For both components, peak amplitude and peak latency were analysed. In addition, the amplitude of a difference wave was calculated by subtracting the Go response from the NoGo response to give an N2d (difference) and a P3d (difference). It is suggested that N2d represents conflict monitoring whilst P3d represents response inhibition (41). Analysis of N2 and P3 amplitude was conducted using a Mixed ANOVA with GROUP (control, OCD) as the between-measures factor and CONDITION (Go, NoGo) and SITE (Fz, Cz, Pz) as within measures factors. P3 latency was also analysed using this method. However, N2 latency was analysed using only GROUP and CONDITION because the amplitude analysis revealed that N2 was only present in both groups at Fz, meaning SITE was not relevant. N2d was analysed using an independent sample t-test for this same reason whilst P3d was analysed using a Mixed ANOVA with GROUP as the between-measures factor and SITE as the within-measures factor.

Hypothesis 2 (within OCD): Rather than conduct one large analysis that included both comorbidity status and symptom cluster we opted to analyse the two separately due to the small samples that would have arisen if combined (e.g. comorbid depression and symmetry cluster N=3). To investigate the effect of cluster on response inhibition the behavioural data were compared using One-Way ANOVA. For ERP responses P3 amplitude and latency were analysed with a Mixed ANOVA with CLUSTER as the between-measures factor and CONDITION and SITE as within measures factors. For N2 amplitude and latency, which is only found at Fz in participants with OCD, a Mixed ANOVA with CLUSTER as the between-measures factor and CONDITION only as a within measure factor was used. P3d was analysed using a Mixed ANOVA with CLUSTER as the between-measures factor and SITE as the within-measures factor whilst N2d was compared using One-Way ANOVA. Comorbidity analysis was conducted in the same way but cluster was replaced with COMORBIDITY as the between-measures factor for all ANOVAs and One-Way ANOVAs were replaced with independent-sample t-tests.

Results

ERP but not behavioural measures differentiate between OCD and control participants

There was no significant difference in the mean reaction time for correct responses between control (Mean ± SD; 339.5 ± 63.0 ms) and OCD participants (354.6 ± 78.4 ms; t (99) = 1.069, p = 0.288; Figure 2A). There were also no differences in the number of omission errors (Control = 2.3 ± 3.0; OCD = 1.9 ± 2.2; t (99) = 0.655, p = 0.514, Figure 2B) or commission errors (Control = 0.9 ± 0.0; OCD = 0.8 ± 0.2; t (99) = 0.415, p = 0.679, Figure 2C) between the two groups.

Grand averages of the ERP responses of the OCD and control groups are shown in Figure 3. Analysis of N2 amplitude revealed a significant main effect of GROUP (F (1, 99) = 5.64, p = 0.019) with the control group having a larger overall N2 component when all sites are considered. There was also a significant main effect of CONDITION (F (1, 99) = 80.12, p < 0.001), with the NoGo condition eliciting a greater N2 response. Finally, there was a significant main effect of SITE (F (2, 198) = 134.91, p < 0.001); pairwise comparisons show all sites differed significantly from each other (p < 0.001 Fz>Cz>Pz). There was no negative response at Pz indicating N2 was not found at this location when both groups are considered together. There was a significant SITE x GROUP interaction (F (2, 198) = 13.22, p < 0.001) with interaction contrasts revealing that N2 was no different between the two groups at Fz, both groups lacked a response at Pz and finally, the Cz response was significantly different due to no appreciable negative response being present in the OCD group. There was no significant GROUP x CONDITION interaction (F (1, 99) = 0.13, p = 0.910). There was a significant SITE x CONDITION interaction (F (2,198) = 26.62, p < 0.001) driven by the difference in Go and NoGo and the fact that N2 is not present at all sites. There was a significant GROUP x SITE x CONDITION interaction (F (2, 198) = 21.88, p < 0.001). Examination of the contrasts revealed this interaction arises because of the more localised N2 in OCD participants and the fact that N2 is greater at the frontal location in the NoGo condition in comparison to the Go Condition.

N2 latency analysis focussed on Fz because this was the only location in which it was present for both groups revealed a significant main effect of GROUP (F (1, 99) = 6.81, p = 0.01) with the OCD group having a significantly larger latency, i.e. a slower response. There was also significant main effect for CONDITION (F (1, 99) = 10.79, p = 0.001) with larger latencies, i.e. slower responses in the Go condition. There was no significant interaction (F (1, 99) = 1.06,

$p = 0.306$). Finally, N2d analysis revealed that the OCD group had a larger difference between the two conditions than the control group (t (99) = 2.37, $p = 0.020$).

Analysis of P3 amplitude found a significant main effect of GROUP (F (1, 99) = 8.43, $p = 0.005$) with the OCD group having a larger P3 amplitude. There was also a significant main effect of CONDITION (F (1, 99) = 113.42, $p < 0.001$), with the NoGo condition eliciting a greater amplitude. Finally, there was a significant main effect of SITE (F (2, 198) = 164.29, $p < 0.001$) with pairwise comparisons showing all sites differed significantly from each other ($p < 0.001$ Cz>Pz>Fz). There was a significant SITE x GROUP interaction (F (2, 198) = 12.56, $p<0.001$). To break this down, interaction contrasts were performed comparing P3 amplitude across the electrode sites and revealed that controls have a larger P3 at Fz, whilst those with OCD have higher P3 amplitudes at Cz and Pz. There was also a SITE x CONDITION interaction (F (2,98) = 149.16, $p < 0.001$). This was driven by the fact that in the Go condition the responses at Cz and Pz were comparable and larger than at Fz, whilst in the NoGo condition, responses were greatest at Cz and comparable and lower at Fz and Pz. Finally, there was a significant GROUP x CONDITION interaction (F (1, 99) = 5.99, $p = 0.016$). Again, examination of the data and interaction contrasts revealed that this interaction was due to a greater P3 amplitude difference between the OCD and controls in the Go Condition compared to the NoGo condition ($p = 0.016$). There was no significant GROUP x SITE x CONDITION interaction (F (2, 198) = 0.45, $p = 0.64$)

P3 latency analysis showed no significant main effect of GROUP (F (1, 99) = 0.00, $p = 0.996$), but there was a main effect of CONDITION (F (1, 99) = 7.57, $p = 0.007$), with larger P3 latencies i.e. slower responses in the Go condition. There was also a main effect of SITE (F (1.54, 151.92) = 78.39, $p < 0.001$). Contrasts revealed that the P3 response was slowest at Fz

($p < 0.001$) and fastest at Pz ($p < 0.001$). There was no significant GROUP x SITE interaction (F (1.54, 151.92) = 1.25, p = 0.283) or GROUP x CONDITION interaction (F (1, 99) = 2.28, p = 0.135). However, there was a significant SITE x CONDITION (F (2,198) = 3.23, p = 0.042) with latencies in the Go condition varying more with SITE than in the NoGo condition. There was a significant GROUP x SITE x CONDITION (F (1.76, 173.89) = 3.23, p = 0.048). Examination of the interaction contrasts revealed that the difference in P3 latency between the Go and NoGo conditions across the OCD and control group differed between Fz and Pz (p = 0.025) and Cz and Pz (p = 0.041). P3d amplitude analysis revealed a significant main effect of GROUP (F (1,99) = 5.99, p = 0.016) with larger responses in the control group. In addition, there was a main effect of SITE (F (2,198) = 159.38, $p < 0.001$), with pairwise comparisons showing significant differences between all electrode sites ($p < 0.001$; Cz>Fz>Pz), indicating the greatest inhibition effect at central locations. There was no significant interaction (F (2,198) = 0.45, p = 0.640).

There were no differences between different symptom clusters

There was no significant difference in the mean reaction time for correct responses between different three clusters (Symmetry = 348.3 ± 104.4 ms; Forbidden Thoughts = 354.4 ± 70.9 ms; Cleaning = 362.3 ± 82.2 ms; F (3,53) = 0.536, p = 0.660). There were also no differences in terms of the number of omission errors (Symmetry = 2.6 ± 2.7; Forbidden Thoughts = 2.0±2.4; Cleaning = 1.6 ± 1.7; F (3,53) = 0.507, p = 0.679) or commission errors (Symmetry = 1.0 ± 1.0; Forbidden Thoughts = 0.5 ± 0.8; Cleaning = 1.1 ± 1.0; F (3,53) = 1.650, p = 0.189).

For N2 amplitude and latency, there was no significant main effect of CLUSTER (Amplitude: F (2, 44) = 0.023, p = 0.977; Latency: F (2, 44) = 0.898, p = 0.415). There was also no main effect of CONDITION on latency (F (1, 44) = 0.092, p = 0.763). However, mirroring the whole cohort analysis, there was a main effect of CONDITION on amplitude (F (1, 44) = 10.241, p

= 0.003) with a greater response in the NoGo condition. There were no significant CONDITION x CLUSTER interactions (Amplitude F (2,44) = 1.78, p = 0.180; Latency F (2,44) = 0.31, p = 0.733). There was no significant difference between the clusters for N2d amplitude (F (2,44) = 0.884, p = 0.420).

For P3 there was no significant main effect of CLUSTER (Amplitude: F (2,44) = 0.23, p = 0.799; Latency: F (2,44) = 0.27, p = 0.767), CONDITION (Amplitude: F (1, 44) = 0.04, p = 0.849; Latency: F (1,44) = 0.13, p = 0.725) or SITE (Amplitude: F (1, 44)=8.60, p = 0.427; Latency: F (1.35, 59.18) = 3.42, p = 0.057). There were also no significant interactions (Amplitude: CONDITION x CLUSTER F (2,44) = 0.31, p = 0.736, CONDITION x SITE F(2,88) = 0.19, p = 0.826, SITE x CLUSTER F (4,88) = 0.47, p = 0.760, CONDITION x SITE x CLUSTER F (4,88) = 1.27, p = 0.287; Latency CONDITION x CLUSTER F (2,44) = 0.54, p = 0.586, CONDITION x SITE F (1.52, 66.77) = 0.94, p = 0.374, SITE x CLUSTER F (4, 88) = 1.27, p = 0.287, CONDITION x SITE x CLUSTER F (3.04, 66.77) = 0.75, p = 0.526). P3d showed no significant main effects (CLUSTER F (2, 44) = 0.309, p = 0.736; SITE F (2, 88) = 0.192, p = 0.826) or interaction (CLUSTER x SITE F (4, 90) = 0.97, p = 0.429).

There were selected effects of comorbid depression on behavioural and ERP responses.

The average reaction time for correct responses on the Go trials did not differ between those with (334.9 ± 66.8 ms) and without comorbid depression (368.6 ± 84.1; t (46) = 1.491, p = 0.143). There were also no differences in terms of the number of commission errors made (OCD 1.1 ± 2.6; OCD with comorbid depression 0.6 ± 1.7; t (46) = 1.622, p = 0.101). However, there was a significant difference for omission errors (t (99) = 2.426, p = 0.019) with those with OCD (2.8 ± 0.2) making more omission errors that those with OCD and comorbid depression (1.3 ± 0.3).

For N2 amplitude and latency, there was no significant main effect of COMORBIDITY (Amplitude: F (1, 46) = 2.70, p = 0.107; Latency: F (1, 46) = 0.061, p = 0.806). However, as with the other comparisons, there was main effect of CONDITION on amplitude (F (1, 46) = 85.98, $p < 0.001$) with bigger N2 responses during the NoGo condition. Again, mirroring the main OCD vs control analysis, there was also a significant main effect of CONDITION on latency (F (1, 46) = 9.46, p = 0.004), with slower responses during the Go conditions. There were no significant CONDITION x COMORBIDITY interactions (Amplitude F (1, 46) = 0.26, p = 0.613, Latency F (1, 46) = 1.78, p = 0.188). For N2d there was no significant difference for amplitude (F (1,46) = 2.19, p = 0.145) between those with and without comorbid depression.

For P3 amplitude there was no significant main effect of COMORBIDITY (F (1, 46) = 0.34, p = 0.562). As with the main comparison between those with and without OCD, there was a significant main effect of CONDITION (F (1, 46) = 29.48, $p < 0.001$, with bigger P3 responses during the NoGo condition. There was a significant main effect of SITE (F (2, 92) = 76.65, $p < 0.001$) with pairwise comparisons revealing that all sites differed significantly from each other ($p < 0.001$; Cz>Pz>Fz) in the same way as the main group comparison. There was also a significant CONDITION x SITE interaction (F (2, 92) = 64.51, $p < 0.001$) following the same pattern as the main group analysis. There were no other significant interactions for P3 amplitude (CONDITION x COMORBIDITY F (1, 46) = 2.72, p = 0.106, SITE x COMORBIDITY F (2, 92) = 0.28, p = 0.757, SITE x CONDITION x COMORBIDITY, F (2, 92) = 1.25, p = 0291).

For P3 latency there was a significant main effect of COMORBIDITY (F (1, 46) = 6.02, p = 0.018), with those with comorbid depression demonstrating faster P3 responses. There was also a main effect of SITE (F (2, 92) = 34.50, $p < 0.001$), pairwise comparisons revealed significant differences between Fz and Cz ($p<0.001$) and Fz and Pz ($p < 0.001$) with the fastest P3 response at Pz and the slowest at Fz as found for the main analysis. There was no significant main effect of CONDITION (F (1, 46) = 1.95, p = 0.169), but there was a significant SITE x CONDITION interaction (F (2, 92) = 18.20, $p < 0.001$) in line with the results from Hypothesis 1. There was a significant CONDITION x COMORBIDITY interaction (F (1, 46) = 8.80, p = 0.005); during the Go condition the OCD only group has a slower P3 latency compared to the OCD with comorbid depression, however during the NoGo condition the groups demonstrate similar latencies for P3. There was no SITE x COMORBIDITY interaction (F (2, 92) = 1.88, p = 0.158). Finally, there was also a significant three-way interaction CONDITION x SITE x COMORBIDITY (F (2, 92) = 5.25, p = 0.007). Contrasts revealed that the group differences across the Go and NoGo differed across the Fz vs Pz (p = 0.004) and Cz vs Pz (p = 0.004).

For amplitude of P3d there was no significant main effect of COMORBIDITY (F (1, 46) = 2.72, p = 0.106), but there was a main effect of SITE (F (2, 92) = 64.52, $p < 0.001$), where contrasts revealed that the inhibition effect of P3d was significantly different between all electrode sites ($p < 0.001$; Cz>Fz>Pz). There was no significant SITE x COMORBIDITY interaction (F (2, 92) = 1.25, p = 0.291).

Discussion

The aim of this study was to investigate response inhibition in OCD using behavioural and ERP measures by examining distinct symptom clusters and the presence of comorbid depression. We set out to test three specific hypotheses; that there will be significant differences

in response inhibition between i) control and OCD participants and ii) OCD participants with different symptom clusters and comorbidity status.

Our OCD cohort consisted of participants identified as belonging to all three of the symptom clusters identified and accepted as part of OCD according to DSM-5 (9, 12). Furthermore, as is commonly found, just over half of our participants reported comorbid depression (13). Together, these features suggest we had an ecological valid cohort. When this cohort was compared as a whole to healthy control participants, we found no differences in behavioural measures of response inhibition, as has been found previously (23-29). As would be expected for ERP components linked to response inhibition, both N2 and P3 showed greater amplitude in the NoGo compared to the Go condition. However, importantly for our first hypothesis, there were significant differences between OCD and control participants. In control participants the N2 component was present at both frontal and central locations, whereas within the OCD cohort, whilst comparable to controls frontally, N2 was absent at the central location, indicating a more localised response in OCD. This site-dependent effect has also been found by others (26, 33) and may have contributed to previous inconsistent results where different electrode sites had been included in analysis or combined in different ways. The OCD participants also had a longer latency response. Previous research has suggested that the latency of the N2 component on this task reflects the speed of the monitoring of conflict, and therefore the increased latency may be indicative of OCD influencing the time course of inhibitory activity by slowing down the speed of response inhibition (45). The slower latency would suggest reduced conflict monitoring in OCD causing slower responding to the occurrence of conflicts. There is some evidence to support deficits in conflict monitoring in OCD (46) However the greater difference in N2 amplitude between the Go and NoGo condition (N2d) found in the present study, suggests overactive conflict monitoring in this cohort (45, 47). The conflicting

findings in the present study are not entirely unprecedented with a recent review suggesting that it is still not clear whether conflict monitoring is reliably altered in OCD (48). Based on the findings presented here there is evidence for slower but greater conflict monitoring. Interestingly, the results for P3 also show this mixed picture. We found that OCD participants exhibited a greater P3, implying greater response inhibition, a finding in line with previous research which also suggests that the increased P3 reflects hyperactivity of the underlying neuronal networks between the orbitofrontal and anterior cingulate cortices and basal ganglia in OCD (31). However, we also found a smaller P3d, indicative of reduced response inhibition (47).

These control versus OCD participant differences, whilst interesting, are not new findings. The critical element of the current study was to investigate whether there were differences between clusters or those with and without comorbid depression, which may have confounded previous studies. The cluster analysis revealed no differences between the clusters on any measure of response inhibition. This indicates that cohorts with different symptom clusters have not contributed to the inconsistent results previously found. However, it is important to acknowledge that whilst the overall sample size for the OCD cohort in the present study is considerably larger than has been found in many previous studies (22, 24, 26, 33, 46, 49), the number of participants in each cluster was limited, especially for the Symmetry cluster.

The comparison of OCD participants with and without comorbid depression revealed that those with OCD only made more errors of omission that those with comorbid depression. This was the only significant difference found in any behavioural measure for this study. Errors of omission can be considered an index of response execution, as opposed to errors of commission, which are an index of response inhibition. The increased level of omission errors

in those with OCD only is indicative of a deficit in sustained attention (50). Whilst the higher level of omission errors reported here contrasts with previous work where OCD (in the absence of comorbidities) was associated with a decrease in omission and an increase in commission errors (22), it is in line with studies showing poorer sustained attention in OCD (51, 52). However, this does not explain why the presence of comorbid depression would effectively protect again errors of omission. Previous research has shown errors of omission in depressed participants are comparable to control participants (53) but there is no evidence to suggest those with comorbid depression somehow have improved sustained attention, although this may be something to consider in future research. Irrespective of this, the present data suggest that this particular deficit in OCD leading to increased errors of omission is not due to the presence of depression as has been previous suggested (54) and more recently discounted elsewhere (55, 56).

There were no significant group differences for most ERP measures but there was a reduced latency for P3 in those with comorbid depression. This is in line with previous studies of depression showing a short latency P3 in patients with depression relative to healthy controls (57). P3 latency is believed to represent the speed of high-level cognitive activity (58) such that a decrease in latency would suggest an increase in the speed of processing during the stimulus-evaluation and decision-making phases of response. These results, as with the findings on errors, imply that the presence of comorbid depression somehow supports improved task performance. It remains to be seen whether this arises because of effective compensation mechanisms or prior treatment of depression (because current treatment was matched in the present study), for example. Whilst the impact on response inhibition reported here is small, the prevalence of this comorbidity is high and, therefore, it can be argued that it is beneficial to differentiate comorbidity in analyses of response inhibition. Given these findings, it is

possible that the presence of comorbid depression could have had a small impact on previous results in response inhibition studies with OCD participants. However, it is important to recognise the limitations of the work presented here. Whilst the sample size for the two groups was satisfactory, we did not independently assess depression.

As well as the limitations of sample size discussed above, it is important to note that this study used only one measure of response inhibition – the Go/No-Go task – and this is a limitation. Response inhibition is not a unitary trait, it involves three distinct elements: action postponement, action restraint and action cancellation (59) and different tasks access different sub-components of response inhibition. The Go/No-Go task may contain response-selection and 'waiting' elements but does not access information related to response cancellation (60). One task that does access this is the stop-signal task (SST), but this in turn does not access the sub-components available from the Go/No-Go task (60). Perhaps unsurprisingly given that they access different subcomponents of response inhibition, performance on these tasks relies on slightly different neural circuitry with the Go/No-Go task highly dependent on the inferior frontal cortex and the SST more reliant on normal functioning of the dorso-medial striatum (61-63). Previous studies with OCD participants have revealed that there are changes in the inferior frontal cortex for regional blood flow (64, 65), grey matter volume (66) and activation during Go/No-Go tasks (67). However, OCD is also linked to changes in cortico-striatal circuitry, albeit with most changes noted for the ventral rather than dorsal striatum (68). Nonetheless this means that using the SST may be a worthwhile future investigation for this clinical group. In addition to only using one task, we only included participants with comorbid depression because this has been shown to be the most comorbidity found in over 50% of individuals with OCD (13). However, there are several other comorbid conditions which are relatively common in OCD including social phobia (35.3%), generalised anxiety disorder

(34.1%) and specific phobia (31.6%). Therefore, these additional comorbidities may also impact on the measures we collected and future studies should consider including a wider range of comorbidities.

Conclusions

In summary, the current study has examined response inhibition in different clinical subgroups within OCD, an important step in revisiting research where the heterogeneity of the condition has been overlooked previously. From the results, we can conclude that symptom cluster is unlikely to have contributed to the previous inconsistencies in findings. However, the presence of comorbid depression may have a small impact on results and, therefore, should be considered for separate analysis in future studies.

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Figures

Figure 1 Timescale of the stimulus presentation of the Visual Continuous Performance Task (VCPT) (A) The stimuli presented for each of the key conditions (B).

Figure 2 There were no differences in the reaction times for correct responses (A), omission (B) or commission errors (C) between the control and OCD group. Due to the low error number, data is shown as mean ± SEM.

Figure 3 Normalised grand averages of OCD (grey) and control groups (black) during Go and NoGo conditions and the NoGo-Go difference waveforms. Dotted lines indicate stimulus onset. The vertical scale bar is 4μV and the horizontal scale bar is 400ms**.**

Tables

Table 1 Summary of participant characteristics matched between OCD (*N* = 48) and control (*N* = 53) participants. All continuous variables are given as mean ± SD. Matching was confirmed with independent sample t-tests (age and years in education) or chi-square test of independence (gender and handedness).

	OCD		Control		Test statistic	Df	p-value
Male	18		18		0.14	1	0.711
Female	30		35				
Handedness Right	26		29		0.27	2	0.873
Handedness Left	17		20				
Handedness Ambidextrous	5		4				
	Mean	SD	Mean	SD			
Age (years)	36.1	11.4	40.3	11.6	1.89	99	0.062
Years in Education	14.8	2.6	15.6	2.7	1.45	99	0.150

Table 2 Summary of participant characteristics matched between distinct OCD symptom clusters (Symmetry *N*=7; Forbidden Thoughts *N*=25, Cleaning *N*=16). All continuous variables are given as mean ± SD. Matching was confirmed with One-Way ANOVA or Chi-square test

	Symmetry		Forbidden Thoughts		Cleaning		Test statistic	Df	p-value
Male	2		11		5		0.96	2	0.620
Female	5		14		11				
Handedness: Right	4		14		8		5.81	4	0.214
Handedness: Left	3		10		4				
Handedness: ambidextrous	0		1		4				
Medication: none	2		12		4		8.85	4	0.065
Medication: antidepressant	2		11		10				
Medication: Other	3		2		1				
No CBT	4		18		14		2.64	2	0.267
CBT	3		7		2				
OCD only	3		9		8		0.79	2	0.673
Comorbid Depression	4		16		8				
	M	SD	M	SD	M	SD			
Age (years)	37.6	9.4	34.1	11.2	38.4	12.5	0.77	2	0.471
Years in Education	13.9	3.8	15.3	2.2	15.5	2.5	1.00	2	0.376
Age of onset (years)	32.3	3.6	26.1	1.91	25.7	8.2	1.48	2	0.239
Illness duration (months)	63.0	65.6	96.7	103.5	153.1	133.4	2.03	2	0.144
Illness Severity	26.4	5.1	23.2	5.4	26.1	5.6	1.89	2	0.163

Table 3 Summary of participant characteristics matched between those with OCD only (*N* = 20) and those with OCD and depression diagnoses (*N* = 28). All continuous variables are given as mean ± SD. Matching was confirmed with independent sample t-tests (continuous variables) or chi-square test of independence (categorical).

	OCD only		OCD + depression		Test statistic	Df	p-value
Male	10		8		1.04	1	0.306
Female	10		20				
Handedness: Right	8		18		3.34	2	0.189
Handedness: Left	10		7				
Handedness: ambidextrous	2		3				
Medication: none	10		8		3.42	2	0.181
Medication: antidepressant	8		15				
Medication: Other	1		5				
No CBT	16		20		0.46	1	0.499
CBT	4		8				
	M	SD	M	SD			
Age (years)	34.0	11.8	37.5	11.0	0.14	46	0.886
Years in Education	14.8	3.1	14.9	2.2	1.00	46	0.376
Age of onset (years)	26.0	1.9	27.5	9.9	0.54	46	0.592
Illness duration (months)	96.7	115.4	120.5	112.2	0.72	46	0.478
Illness Severity	24.3	5.7	24.9	5.5	0.34	46	0.736